A STUDY ON THE PREVALENCE AND RISK FACTORS ASSOCIATED WITH PERIPHERAL VASCULAR DISEASE IN TYPE2 DIABETES MELLITUS.

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MADURAI MEDICAL COLLEGE, MADURAI

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON THE PREVALENCE AND RISK FACTORS ASSOCIATED WITH PERIPHERAL VASCULAR DISEASE IN TYPE2 DIABETES MELLITUS"** submitted by **Dr.E.SUBBIAH** to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment for the award of Doctor of Medicine, is a bonafide work carried out by him under my guidance and supervision. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other universities.

Prof. Dr.S. VADIVEL MURUGAN, M.D.,
Additional Professor of Medicine
Department of Medicine,
Madurai Medical College,
Madurai

Prof. Dr.A. AYYAPPAN.M.D.
Professor and head,
Department of medicine
Madurai Medical College
Madurai

DECLARATION

I**, Dr.E.SUBBIAH, solemnly** declare that the dissertation titled **"A STUDY ON THE PREVALENCE AND RISK FACTORS ASSOCIATED WITH PERIPHERAL VASCULAR DISEASE IN TYPE2 DIABETES MELLITUS"** has been prepared by me.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of MD Degree Branch I (General Medicine)

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: Madurai **Dr.E.SUBBIAH**
Date:

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ABBREVIATIONS

1	PVD	-	Peripheral Vascular Disease
2	DM	-	Diabetes Mellitus
3	HT	-	Hypertension
4	CAD	-	Coronary artery Disease
5	CEVD	-	Cerebro Vascular Disease
6	ABI	-	Ankle – Brachial Index
7	LDL	-	Low-Density Lipoprotein Cholesterol
8	HDL	-	High – Density Lipoprotein Cholesterol
9	TGL	-	Serum Triglycerides
10	FPG	-	Fasting Plasma Glucose
11	BMI	-	Body Mass Index
12	WHR	-	Waist – Hip Ratio

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INTRODUCTION

Peripheral vascular disease (PVD) is one of the significant Macro vascular complication of type 2 diabetes mellitus. Peripheral vascular disease assumes importance, for prevention of morbidity and mortality related to diabetic foot. PVD is the single most important factor which raises the incidence of leg amputation risk in type 2 DM patients. While peripheral neuropathy alone leads to, tropic ulcer which has an chronic indolent course provided super added infection is prevented and controlled, the presence of PVD leads to ischaemic foot which leads to rapid spread of infection, by means of two mechanisms.

I) Due to decreased supply of oxygen, nutrients and decreased accessibility of neutrophils and immune effectors to the area of injury and infection.

II) Decreased antibiotic accessibility to the affected area due to ischaemia.

Further the importance of PVD assessment assumes importance in the light of a long duration of asymptomatic period in the natural course of the disease. Further the avenues for

development of claudication symptoms of PVD in today's fast changing sedentary life style of the people are restricted, and hence the importance of active screening for the same with special emphasis on ankle – Brachial index gains importance for saving the limbs of Diabetic patients.

REVIEW OF LITERATURE

Definition:

PVD:

Peripheral vascular disease is defined as signs, symptoms or abnormal non-invasive tests in one or both legs attributable to obstructive atherosclerotic disease.

CLI:

Critical limb ischaemia is defined as chronic ischemic rest pain attributable to objectively proven arterial occlusive disease.

PREVALENCE:

Several population studies have shown that diabetes is a strong risk factor for PVD (4). In both Diabetic and non diabetic subjects the prevalence of PVD increases markedly with advancing age. In several studies PVD was found in approximately 20% of the diabetic patients (5). In the united kingdom prospective diabetes study (**UKPDS**) PVD was found in 11% of patients after a follow up period of 6 years from the time of diagnosis of type 2 diabetes (6). It can be safely concluded based on these population studies, that PVD is a highly prevalent disorder, in particular in the elderly diabetic patient. Unfortunately, the awareness of the physicians that these patients have PVD is low, resulting

in inadequate preventive measures for reducing lower extremity amputation and to reduce the grossly elevated general cardiovascular risk (7, 8).

SYMPTOMATIC AND ASYMPTOMATIC PVD:

Several epidemiological studies and also in the **UKPDS** study symptoms of claudication were reported in only about 25% of the patients with an ABI of <0.8, indicating that for each patient with claudication there are three patients with silent PVD(6). The reason for asymptomatic PVD could be due to concomitant neuropathy and many elderly patients do not walk far enough to experience symptoms of intermittent claudication.

THE FATE OF LEG:

In diabetic patients the atherosclerotic changes in PVD seem to be more extensive and also more aggressive, resulting in faster progression of disease. In diabetic patients with critical limb ischemia, the risk of limb loss is markedly increased, gangrene being reported to develop in 40% of such patients (9). Diabetes is the leading cause of non traumatic lower limb amputation and the major reasons for a lower extremity amputation are PVD and / or a deep infection [8,10,11].

THE FATE OF THE PATIENT WITH PVD:

PVD is closely associated with occlusive disease in other arterial territories and it is a strong predictor of future cardiovascular morbidity and

mortality (9). Mortality is increased in patients with PVD in parallel to the severity of the disease (12). In one study approximately 20% of the patients with intermittent claudication and diabetes, had died after two years of follow up (2, 12). The poorest out come is observed in diabetic patients who have undergone a major amputation.

SITES OF PVD:

The anterior tibial, posterior tibial followed next by the peroneal arteries are the common arteries occluded in type 2 diabetic subjects.

Involvement of deep formal artery and arteries below the knee is the common site of PVD in diabetic subjects with relative sparing of arteries of foot, rendering these distal segments suitable for byepass surgery (19,39). Why atherosclerosis in diabetic subjects preferably affects lower leg below knee arteries, is at present not well under stood. It is important to realize that foot vessels Dorsalis pedis, plantar and tarsal arteries are spared usually in diabetes subjects with PVD (2).

Strandness and co workers (21) performed a blinded histological review of amputation specimens prepared with periodic acid schiff staining and demonstrated no difference between diabetic and non diabetic specimens. Both groups had similar patterns of atherosclerosis with a paucity of occlusive disease at the arteriolar level. Barner and associates (22) measured the

femoro-popliteal bye-pass grafts blood flow rates as a measure of vessel reactivity after infusion of papaverine in to the patients vascular out flow bed and they recorded no difference between diabetes and non diabetics. These studies have reinforced the newer theory that a unique small vessel disease does not exist in diabetic foot patients. In the past based on the false presumption that small vessel disease at the level of small foot arteries and arteriolar level, diabetics were not treated as aggressively with revascularization as is now standard.

RISK FACTORS :

Hyperglycemia

Hyperglycemia seems to be a stronger risk factor for PVD (6) .In **UKPDS**, each 1% increment in HbA1c was associated with a 28% increase in the risk for PVD (6). **Smoking** is also a stronger risk factor for PVD (6, 23). Both hyperglycemia and smoking can result in the formation of advanced glycation end products, and in enhanced oxidative stress within the vessel wall, resulting in development and progression of PVD.

The Framingham offspring study examined 1554 males and 1759 females for PVD. In this population based study the odds ratio for PVD was 2.3 among diabetic vs non diabetic patients (13).

Smoking

The Framingham heart study found a strong relationship between the number of cigarettes smoked and the incidence of intermittent claudication and a multivariate analysis identified smoking as a strong single risk factor for development of symptomatic obstructive arterial disease(16).The occurrence of intermittent claudication is twice as frequent in smokers as non smokers. In the EDINBURG artery study PVD prevalence strongly and positively related to life time cigarette smoking (17).

Dyslipidaemia

Diabetic PVD is associated with an atherogenic dyslipidaemia, which is characterized by elevated triglycerides, low level of HDL cholesterol and increased number of small dense LDL particles and elevated LP(a) (9,5,6,24).

The association of hypercholesterolaemia with atherosclerosis of the lower extremities has been known for 60 years. The Edinburgh artery study reported a higher prevalence of PVD in association with higher serum cholesterol and lower HDL cholesterol in multiple logistic regression analysis (17).

The cardiovascular health study reached similar conclusions among its sample of 5084 subjects aged 65 years or older with PVD defined as an, ABI less than 0.9 (18).

AGE AND MALE SEX

Older age is also an important risk factor for PVD in patients with diabetes (9, 25). As age advances there is a linear rise in risk of PVD. In the Edinburgh artery study the prevalence of claudication and PVD increased as the age advances.

Although it has been said that men are affected with symptomatic PVD between two to five times as frequently as women (14) in the meta analysis review of meijer et al a two fold or higher prevalence of claudication among men was seen in only one of the 13 studies (15).

HYPERTENSION

The cardio vascular health study reported about a 50% higher prevalence of an ABI less than 0.9 associated with hypertension in a multivariate analysis adjusted for age, smoking, Diabetes and dyslipidaemia(18).

Novel risk factors like hyperhomocystineamia have also been proposed to operate in development of PVD (26).

RELATIONSHIP OF CORONARY ARTERY DISEASE (CAD) AND CEREBROVASCULAR DISEASE WITH PVD:

Atherosclerosis, the underlying cause of PVD being a multi factorial progressive condition involves multiple vascular beds. Therefore it is not surprising that patients with PVD often have extensive CAD and cerebro vascular disease. The prevalence of CAD among persons with claudication in the general population is between two to four times higher than in non-claudicants. Around 50% of claudicants suffer from angina, whereas patients with angina are six times more likely to have claudication (19).

Both claudication and asymptomatic PVD in the Edinburgh artery study population were significantly, associated with greater intima media thickness of the carotid arteries as assessed by ultrasound (20).

DIABETIC NEPHROPATHY AND PVD :

Diabetic nephropathy individuals have an increased incidence of PVD, and the disease has a more severe course in these patients.

The diabetic nephropathy patients have an high risk of development of PVD since nephropathy leads to generalized acceleration of atherosclerosis in all regional vascular beds.

ABNORMAL MICROCIRCULATION AND PVD:

Microangiopathy of tissues, and heamorrheological abnormalities play a lesser role in diabetic foot. Because of sensory neuropathy, a patient with severe ischemia may have no rest pain and because of autonomic neuropathy, subcutaneous A-V shunt blood flow is increased, resulting in a relatively warm skin, thereby falsely reassuring the clinician. Hence the importance of active screening for PVD becomes all the more important in type 2 diabetic subjects.

DIAGNOSIS OF PVD :

All diabetic patients routinely should undergo clinically palpation of pedal pulses, dorsalis pedis and posterior tibial artery. In all diabetics over the age of 50 and those who have symptoms of intermittent claudication, or foot ulcer, should have their ankle-brachial pressure index recorded for both legs separately. An ABI of less than 1 is abnormal. The various investigative modalities for diagnosis of PVD are discussed below

RECORDING OF ANKLE-BRACHIAL INDEX :

Ankle-brachial pressure index is recorded using a sphygmomanometer and hand held Doppler.

$$\text{ABI} = \frac{\text{SYSTOLIC PRESSURE IN ANKLE}}{\text{SYSTOLIC PRESSURE IN ARM}}$$

RECORDING OF TOE-PRESSURES :

Toe systolic pressure is measured in the hallux using a small cuff (2cm) and photoplethysmography or laser Doppler fluxmetry. The toe pressure is normally approximately 10 mmhg less than the ankle pressure. Toe arteries are affected by media calcification to a lesser extent. For screening purpose a toe systolic blood pressure index of >0.60 can be interpreted as normal.

Transcutaneous Partial Pressure of Oxygen (Tcpo2) – The tcpo2 can be measured with a heated oxygen sensitive probe, which is placed on the dorsum of foot. Several studies have shown that tcpo2 values can identify patients at risk for foot ulceration. Normally values are usually > 60 mmhg. However, it should be noted that various systemic factors such as hypoxia or local factors such as edema and inflammation can affect the measurement, resulting in falsely low values.

DUPLEX SCANNING:

The Doppler ultrasonic flow detector with the combination of real time B-mode scanning in colour and Doppler spectral analysis, referred to as duplex scanning, is the most accurate non invasive method of detecting and following vascular lesions. In addition to providing an image of vascular lesions, it also permits an estimation of blood flow.

Doppler pulse velocity wave forms :

TRIPHASIC FLOW :

There is a rapid forward flow component during systole, a transient flow reversal during early diastole and a slower forward flow component during late diastole. This is the normal triphasic flow.

Biphasic flow :

Losing the forward flow component during late diastole

Monophasic flow :

Wave form is dampened, the systolic peak is lowered and the rate of rise is delayed.

Digital subtraction angiography (DSA)

The gold standard in the evaluation of diabetic patients with PVD is digital subtraction angiography. Here the images of conventional angiography have been greatly enhanced with the advent of this technique, which utilizes subtraction of the bone and soft tissue components to provide better visualization of the contrast column inside the vessels.

BACKGROUND AND PURPOSE OF THE STUDY

Although Diabetic Peripheral Neuropathy and Peripheral Vascular Disease (PVD) both contribute to the development of diabetic foot, the risk of amputation raises steeply only if there is associated PVD (2).

Once there is presence of PVD in an individual, the time duration taken up for development of gross limb and life threatening sepsis after an injury is very rapid and interventions to augment peripheral blood flow during such an acute active sepsis is not possible always. Hence the importance lies in identifying the risk factors responsible for the development of PVD in type 2 diabetes, and in active screening for PVD in all diabetic subjects. Effective implementation of preventive measures in the targeted high risk group will pave the way for Limb protection. Moreover PVD is a sign of severe generalized atherosclerosis (especially CAD), which should be treated aggressively.

Diabetes predominately leads to PVD in the infrageniculate arteries and the risk factors associated with development of PVD in diabetes are found to be advancing age, uncontrolled hyperglycemia, hypertension, dyslipidaemia and smoking.

AIM OF THE STUDY

1. To investigate the prevalence of PVD among type 2 diabetes patients.

2. To assess the risk factors associated with development of PVD.

3. To correlate the prevalence of cardiovascular risk factors and vascular complications of type 2 diabetes with abnormal ABI (ABI<1).

MATERIALS AND METHODS

112 randomly chosen type 2 diabetic subjects attending our Government Rajaji Hospital, Diabetology department OP between June 2008 to November 2008 have formed the study sample. Patients who have other potential causes for PVD other than diabetes related factors were excluded from the study. The exclusion criteria used were

1. Clinical evidence of thromboangitisobliterans.
2. Suspected arteritis subjects.
3. Patients suffering from hypercoagulable states including heamotologic diseases.
4. Hypothyroidism
5. Collagen vascular disorders
6. Valvular heart disease

STUDY DESIGN:

Cross sectional observational analytical study.

Patients were interviewed with special note on elicitation of history regarding symptoms of PVD, in the form of intermittent claudication, Ischeamic rest pain, history of foot ulcers in the past and present. Smoking was recorded in pack-years of cigarettes smoked.

Duration of diabetes since diagnosis was recorded and they were grouped accordingly. The anthropometric measurements were recorded and the body-mass index was calculated. Waist-hip ratio was also recorded.

The blood pressure was recorded in both the upper limbs and both the lower legs using the standard B.P cuff. Hypertension was detected if the upper limb BP was 140/90 or above as per JNC VII norms and also if they are already documented to be hypertensive or on anti-hypertensive medications. If patient is a known hypertensive, the duration of hyper tension since diagnosis was also noted.

An comprehensive physical examination was done and findings recorded. Special importance was given to foot examination. Peripheral neuropathy assessment was done using simmel-weiss monofilament testing and timed vibration sense perception recordings. Symptoms of peripheral neuropathy were also noted.

METHOD OF PERIPHERAL VASCULAR DISEASE DETECTION AND ASSESSMENT

RECORDING OF ANKLE-BRACHIAL INDEX (ABI) :

The patient is placed in the supine position, a cuff, usually the same as used for the brachial pressure and connected to a sphygmomanometer is applied to the lower calf just above the ankle. The hand held Doppler probe

(5-8 MHz) is positioned over the posterior tibial artery and the cuff is rapidly inflated to a pressure sufficient to arrest blood flow. Subsequently the cuff is deflated slowly and the cuff pressure at which pulsatile flow returns is determined. Similar recording is done for dorsalis pedis artery also. The higher of the two recording is the ankle systolic pressure.

ABI is calculated by dividing this ankle pressure by the Doppler pressure measured similarly in the right brachial artery (2, 27, 6). The two consecutive readings were taken. The value for the worse leg was taken in to account.

An ABI >= 1 is normal and if it is between 1 to 0.8 indicates milder form of peripheral vascular disease and an ABI <0.8 to >0.6 is categorized as moderately severe PVD, and most of these patients will have single segment occlusion (9, 28, 29). ABI values below 0.6 indicate critical limb ischemia and usually indicate severe multi- segment disease of peripheral arteries. Limitations of ABI is that the diabetic patient may have media calcification of the arteries of the lower leg which may be less compressible and incompressible arteries, thus a normal value does not exclude a diagnosis of PVD. An ABI >=1.15 suggests that the ankle pressure is falsely elevated (27).

In spite of this limitation, still ABI remains the best screening procedure for PVD in diabetes subjects as proposed by the American diabetes association.(2, 3).

In this present study patients who had an ABI of >=1.15 were screened with duplex color Doppler imaging for screening for PVD, to overcome this limitation.

Screening for Diabetic Nephropathy was done by means of screening for proteinuria using urine dipstick, and renal parameters of blood urea and serum creatinine were estimated. Urine deposits were done to exclude urinary tract infection as a cause of proteinuria. Patients who were initially classified as diabetic nephropathy, had their diagnosis confirmed with corroboration of fundus opinion of Opthalmologist and renal size imaging by USG abdomen and pelvis.

Screening for Diabetic Retinopathy was done by subjecting all the study subjects for an indirect opthalmoscopy at our government rajaji hospital opthalmology department. Patients were categorized under three heads, viz.., no retinopathy, non-proliferative diabetic retinopathy and proliferative diabetic retinopathy using ETDRS criteria. (ETDRS - Early Treatment in Diabetic Retinopathy Study Criteria).

Screening for Coronary Artery Disease was done with details of previous medical records and fresh resting E.C.G**.** Patients who have a history suggestive of angina but with inconclusive resting E.C.G., had been referred to department of cardiology, and they were classified as per the investigations echocardiogram, treadmill exercise ECG and the expert opinion of the cardiologist.

Screening for Cerebro Vascular Disease was done by examination for diminished unequal carotid pulse with bruite on auscultation and prior transient ischeamic attacks and strokes were taken in to account.

Hyperglycaemia and dyslipidaemia was assessed by estimating the FPG and fasting serum lipid profile of the study subjects after an 10 hours fasting.

Serum total cholesterol, HDL-cholesterol and TRIGLYCERIDES were directly biochemically estimated and LDL-cholesterol was calculated as per freedwall's equation.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2002).**

Using this software, range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND OBSERVATIONS OF THE STUDY

PREVALENCE OF PERIPHERAL VASCULAR DISEASE

The total number of cases studied were 112. The prevalence of peripheral vascular Disease, as screened by Doppler ankle Brachial systolic pressure index was 17.9% (n = 20) (Table 7).

Age Distribution of PVD

The age of patients studied ranged from 31 years to 80 years with a mean age of 58.8 years + / - 10.5 S.D. fairly depicting the age group of type 2 diabetic subjects.

As age advances there is a progressive rise in prevalence of peripheral vascular disease. As each decade of life advances, there is a linear and progressive rise in prevalence of peripheral vascular disease with maximum prevalence in the geriatric age group. (Table 10) The mean age of the group with PVD is 58.9 years + / - 11.3 S.D. The progressive rise in prevalence of PVD with advancing age was statistically significant with a P value of 0.0148.

Sub – group analysis among PVD patients showed that as age progresses there was also progressive increase in severity of PVD which was evident by the progressive decline in ABI values with advancing age (P = 0.0021), (Table 13).

SEX DISTRIBUTION

Males constituted 58% (n = 65) and females 42% (n = 47) of the study group (Table 2), Prevalence of PVD was 20% among the males (n = 13) and 14.9% among the females (n = 7) (Table - 9). But the male sex preponderance was not statistically significant (P = 0.6553).

There was no correlation between sex and severity of progression of PVD, as indicated by mean ABI values comparison. (Table - 14). (P = 0.2495).

BODY MASS INDEX (BMI) AND WAIST HIP RATIO (WHR) DISTRIBUTION

BMI and waist Hip ratio was calculated for all patients. The BMI ranged from 19 to 33.2 with a mean of 24.8 + / - 2.3 S.D. The waist hip ratio ranged from 0.7 to 1.16 with a mean of 0.87 + / - 0.09 S.D.

Sub – group analysis revealed that the sub group affected with PVD had statistically significant higher BMI, than non – affected subjects (P = 0.0221) (Table 10). Similarly the sub – group with PVD had higher waist hip ratio values which was also statistically significant (P = 0.0014) (Table 10).

DURATION OF DIABETES MELLITUS AND PVD

The duration of diabetes ranged from newly detected to 28 years, with a mean duration of 8.2 years + / - 6.24 S.D. For patients affected with PVD, the duration of diabetes was found to be longer than others. The mean duration of diabetes for the PVD sub group is 11.47 years + / - 7.58 S.D. While for the rest of the study group it is 7.49 years + / - 5.72 S.D. which was statistically significant. (P = 0.0304) (Table 10).

FASTING PLASMA GLUCOSE (FPG)

Fasting plasma glucose was Bio- chemically analyzed for all the subjects. And it ranged from 88 – 290mg% with a mean FPG of 155.7 mg % + /- 36.9 S.D.

The control of diabetes was relatively poor among the subjects with PVD, when compared with the rest of the study group. The mean FPG for the PVD sub group is 173.6mg % + / - 34.9 SD, while for the other

subjects the mean FPG is 151.8 mg % + / - 36.3 SD and the difference was statistically significant. (P = 0.006) (Table 10).

HYPERTENSION AND PVD.

Prevalence of hypertension was 50.9 % (n = 57) among the study group, which correlates with established fact.

Among the hypertensive diabetic subjects (n=57), 26.3% had peripheral vascular disease and the prevalence of PVD among normotensive diabetes subjects (n = 55) was 9.1% (P = 0.0329) (Table17).

Hypertension raised the risk of PVD, nearly three fold among type 2 diabetic patients. Duration of hypertension among hypertensive diabetic subjects ranged from newly detected to 28 years with a mean duration of 7.18 years + / - 6.04SD.

Among the hypertensive diabetic subjects, the mean duration of hypertension among PVD subjects was 10.83 year + / - 7.79 SD, which was significantly higher than the non – PVD sub group mean of 5.87 year + / - 4.75 SD (P = 0.0165) (Table 10).

SMOKING

41.5% of male patients (N = 27) were smoking in the study group. The duration of smoking ranged from 5 - 35 pack years. There was no smoking in the female sub – set.

Smokers had more than four fold increased risk of having PVD compared with non – smokers.

ABI was less than I in 37.04% of the smokers (n = 10) and whereas it was 7.89% among the non – smoking males. (n = 3), which was statistically significant (P = 0.0098) (Table 12).

There was a positive linear correlation between the duration of smoking (In pack - years) and risk of development of PVD (Table 15).

HYPERTENSION AND SMOKING

Multivariate analysis showed a significant higher risk of PVD among smoking Hypertensive Diabetic males, compared to non smoking Hypertensive Diabetic males.

Smokers among Hypertensive Diabetic males had 58.3% prevalence of PVD (ABI < 1) compared to 14.3% among non – smoking Hypertensive Diabetic males (Table 16) (P=0.0164).

Thus Smoking is an strong additional risk factor for peripheral vascular disease in hypertensive individuals.

Similarly, in the PVD sub – group the mean ABI of Normotensive smokers was 0.85 +/- 0.12 S.D, while that of hypertensive smokers was 0.58 +/- 0.15 S.D. (P=0.0304) (Table 20).

Thus, the presence of hypertension in a smoker, significantly increases the risk of peripheral vascular disease.

Hence, it could be inferred that the addition of smoking is an independent strong risk factor, both in normotensive and hypertensive Diabetic subjects and the risk raises significantly when smoking is associated with hypertension.

DYSLIPIDAEMIA AND PVD

Fasting serum lipid profile was analysed in all the subjects. The LDL – cholesterol ranged from 85Mg% to 164mg% with a mean of 124.2mg % + / - 16.2 SD. HDL cholesterol ranged from 25mg% to 64Mg% with a mean for 33.9 % + / - 6.1 SD. Serum triglycerides ranged from 84mg% to 302mg% with a mean of 164.9mg %+ / - 35.8 SD.

Mean LDL – cholesterol values in the PVD – sub group was 136.1 mg + / - 10.4 SD compared to 121.6mg% + / - 16.2SD in the non – PVD sub – group (P = 0.0001). HDL Cholesterol mean value in the PVD sub – group was 30.5 mg % + / - 4.7 SD compared to 34.7mg % + / - 6.1 SD in the non – PVD group. (P = 0.0009).

Similarly mean triglycerides value in the PVD sub – group was 197.5 Mg% +/-43.7SD compared to 157.8mg% + / - 29.7 SD in the non – PVD group. (P = 0.0001) (Table 11)

Difference in the lipid parameters above were all statistically significant. This shows that individuals with a triad of increased LDL – cholesterol, lower HDL – cholesterol (Higher LDL / HDL ratio) and increased triglycerides were more at higher risk for peripheral vascular disease.

SYMPTOMATIC AND ASYMPTOMATIC PVD

Symptoms of PVD in the form of intermittent claudication, foot ulcers past and present, were present in 50% of the patients with PVD (ABI <1) (Table 19)

One patient presented with intermittent claudication symptoms, but the ABI was more than 1, the patient was referred for further investigations to vascular surgery department, in the form of post – exercise recording of ABI, duplex Doppler scanning of lower limb vessels, x-ray of legs for screening of extensive moncke bergs medial calcification of arteries.

Interesting observation to note is that patients with mild PVD (ABI > 0.8 to < 1), had relatively no symptoms, and as the ABI values decline, there was a trend towards development of symptoms. But, however, there were also asymptomatic patients even in the severe PVD group.

Thus, it is evident that while due importance to be given for symptoms of PVD, since a significant majority of patients are asympomatic it is imperative that all Diabetic subjects be actively screened for PVD with objective measurement of ABI, for identifying

and implementing preventive strategies for foot protection and general cardio vascular protection.

CORONARY ARTERY DISEASE (CAD) AND PVD

The prevalence of CAD in the study group was 20.5% (n=23). Coronary artery disease was present in 60% of the patients with abnormally low ABI (ABI <1) (n=12) compared to 12% among patients with ABI in the normal range (n=11) (Table 18). This shows that patients with peripheral vascular disease, as defined by an ABI of < 1, significantly have higher prevalence of coronary atherosclerosis, and an ABI of < 1 is to be considered as an coronary artery disease equivalent; and ABI < I is an good marker of silent CAD, in type 2 Diabetic subjects.

More over analysis revealed 52.2%of coronary artery disease patients also had evidence of PVD in the form of ABI <1, compared to 9% among non – CAD subjects.

NEPHROPATHY AND PVD

Nephropathy was present in 7.1% of type 2 diabetes patients. Among the patients with nephropathy, there was a high prevalence of PVD to the extent of 75% (Table 18)

Thus, its evident that development of Diabetic Nephropathy Significantly increases the risk of development of co – morbid PVD, since nephropathy leads to generalized acceleration of atherosclerosis in all the regional vascular beds.

DIABETIC RETINOPATHY AND PVD

The prevalence of Diabetic retinopathy was 23.2% in the study group. Among patients with Diabetic retinopathy, 19% had proliferative diabetic retinopathy.

Among the patients with Diabetic retinopathy, 42.5% (N=11) had PVD, compared to 9% among the others. (P=0.0006 – Significant) (Table18)

CEREBRO VASCULAR DISEASE AND PVD

Cerebro vascular disease was present in 6.3% of the study group. Among the patients with cerebro vascular disease 71.4% (n=5) had co – morbid PVD, compared to 14.1% prevalence of PVD among the rest. (P= 0.0019) (Table 18)

25% of the patients with PVD had concomitant co- morbid cerebro vascular disease (n=5) compared to 2.17% prevalence of cerebro vascular disease among the non – PVD sub set.

TABLES & CHARTS

A. CHARACTERISTICS OF CASES STUDIED.

Table 1 : Age Distribution

Age group in years	Cases	
	No	%
31-40	12	10.7
41-50	30	26.8
51-60	37	33
61-70	28	25
Above 70	5	4.5
Total	112	100
Range	31-80 yrs	
Mean	53.8 yrs	
S.D.	10.5 yrs	

Table 2 : Sex Distribution

Sex	Cases	
	No	%
Males	65	58
Females	47	42
Total	112	100

Table 3 : Other quantitative parameters

Parameter	Range	Mean	SD
BMI	19-33.2	24.8	2.3
WHR	0.7 – 1.16	0.87	0.09
Duration of DM (in years)	1-28	8.2	6.24
Duration of HT (in years)	1-28	7.18	6.04

Table 4 : Fasting Plasma Glucose and Lipid Profile parameters

Parameter	Range in mg%	Mean	SD
F.P. Glucose	88-290	155.7	36.9
LDL	85-164	124.2	16.2
HDL	25-64	33.9	6.1
TGL	84-302	164.9	35.8

Table 5 : Other Qualitative Parameter

Parameter	Cases	
	No	%
Smoking among males (65)		
Smokers	27	41.5
Non Smokers	38	58.5
Hyper Tension		
Present	57	50.9
Absent	55	49.1
Symptoms of PVD		
Symptomatic	11	9.8
Asymptomatic	101	90.2
PVD		
Present	20	17.9
Absent	92	82.1
CEVD		
Present	7	6.3
Absent	105	93.8
CAD		
Present	23	20.5
Absent	89	79.5
Nephropathy		
Yes	8	7.1
No	104	92.9
Fundus		
Normal	86	76.8
Abnormal	26	23.2
Neuropathy		
Yes	86	76.8
No	26	23.2

Table 6 : Severity of PVD

Severity of PVD	Cases	
	No	%
Moderate (ABI – 0.6 to <1)	15	13.4
Severe (ABI < 0.6)	5	4.5
Normal (ABI >1)	92	82.1
Total	112	100

Table 7 : Prevalence of PVD

Prevalence of PVD	Cases	
	No	%
Total cases studied	112	100
PVD Cases	20	17.9

B : Association of Risk factors with development of PVD

Table 8 : Age and PVD

Age Group	PVD			
	Present		Absent	
	No	%	No	%
31-40 (12)	1	8.3	11	91.7
41-50 (30)	3	10	27	90
51 – 60 (37)	5	13.5	32	86.5
61 – 70 (28)	9	32.1	19	67.9
Above 70 (5)	2	40	3	60
Mean Age	58.9 yrs		52.7 yrs	
S.D.	11.3 yrs		10.0 yrs	
'p'	**0.0148 Significant**			

Table 9 : Sex and PVD

Sex	PVD			
	Present		Absent	
	No	%	No	%
Males (65)	13	20	52	80
Females (47)	7	14.9	40	85.1
'p'	0.6553 Not Significant			

Table 10 : Relationship between other Quantitative Variables and PVD

Variable	Value for Cases with PVD				'p'
	Present		Absent		
	Mean	SD	Mean	SD	
BMI	25.33	1.67	24.64	2.37	**0.0221 Significant**
WHR	0.92	0.08	0.86	0.1	**0.0014 Significant**
Duration of DM (in years)	11.47	7.58	7.49	5.72	**0.0304 Significant**
Duration HT (in years)	10.83	7.79	5.87	4.75	**0.0165 Significant**

Table 11 : Relationship between FPG, Dyslipidaemia and PVD.

Variable	Value for Cases with PVD				'p'
	Present		Absent		
	Mean	SD	Mean	SD	
F.P. Glucose	173.6	34.9	151.8	36.3	**0.006 Significant**
LDL	136.1	10.4	121.6	16.2	**0.0001 Significant**
HDL	30.5	4.7	34.7	6.1	**0.0009 Significant**
TGL	197.5	43.7	157.8	29.7	**0.0001 Significant**

Table 12 : Relationship between Smoking among males (65)and PVD

Variable	Cases				'p'
	With PVD		Without PVD		
	No	%	No	%	
Smokers (27)	10	37.04	17	62.96	**0.0098 Significant**
Non Smokers (38)	3	7.89	35	92.11	

C : STRENGTH OF ASSOCIATION BETWEEN PVD AND OTHER VARIABLES

Table 13 : Age and severity of PVD

Severity of PVD	Age in years	
	Mean	**S.D.**
Mild	44.8	7
Moderate	63.7	5.1
Severe	67	8.1
'p'	**0.0021 Significant**	

Table 14 : Sex and severity of PVD

Severity of PVD	Sex			
	Male		**Female**	
	Mean	**SD**	**Mean**	**SD**
Mild ABI 0.80 < 1	4	66.7	2	33.3
Moderate ABI 0.6 to 0.8	5	55.6	4	44.4
Severe ABI < 0.6	4	80	1	20
ABI Mean S.D.	0.67 0.18		0.75 0.14	
'p'	0.2495 Not Significant			

Table 15 : Relationship between pack-years of smoking and PVD.

Pack-years of smoking	Total No.Of. Patients	PVD			
		Present		Absent	
		N	%	N	%
5-10 pack years	7	2	28.6%	5	71.4%
11-15 pack years	7	2	28.6%	5	71.4%
16-20 pack years	11	5	45.5%	6	54.5%
> 25 pack years	2	2	100%	0	0%

Table 16 : Association of Hypertension and Smoking with PVD

Among patients with Hypertension(33)	PVD			
	Present		Absent	
	No.	%	No.	%
Smokers (12)	7	58.3	5	41.7
Non smokers (21)	3	14.3	18	85.7
'p'	**0.0164 Significant**			

Table 17 : Relationship between hypertension and PVD.

Variables	Cases				'p'
	With PVD		Without PVD		
	No.	%	No.	%	
Hypertension					
Present (57)	15	26.3	42	73.7	**0.0329 Significant**
Absent (55)	5	9.1	50	90.9	

Table 18 : Relationship between diabetic complications and PVD.

Variables	Cases				'p'
	With PVD		Without PVD		
	No.	%	No.	%	
CEVD					
Present (7)	5	71.4	2	28.6	**0.0019 Significant**
Absent (105)	15	14.1	90	85.7	
CAD					
Present (23)	12	52.2	11	47.8	**0.0001 Significant**
Absent (89)	8	9	81	91.0	
Nepropathy					
Present (8)	6	75	2	25	**0.0004 Significant**
Absent (104)	14	13.5	90	86.5	
Fundus					
Abnormal (26)	11	42.5	15	57.7	**0.0006 Significant**
Normal (86)	9	10.5	77	89.5	
Neuropathy					
Present (86)	18	20.9	68	79.1	0.1524 Not Significant
Absent (26)	2	7.7	24	92.3	

Table 19 : Symptoms of intermittent claudication and PVD.

Variables	Cases				'p'
	With PVD		Without PVD		
	No.	%	No.	%	
Symptoms					
Symptomatic (11)	10	90.9	1	9.1	**0.0001 Significant**
Asymptomatic 101)	10	9.9	91	90.1	

Table 20:

SMOKERS			
Hypertensive smoker		**Normotensive smoker**	
Mean ABI	**S.D**	**Mean ABI**	**S.D**
0.58	$\pm$ 0.15	0.85	$\pm$ 0.12
P= 0.0304. Significant			

DISCUSSION

PREVALENCE

Prevalence of peripheral vascular disease in this study was 17.9% which was comparable to the prevalence as described in other studies. Meijer etal in a meta analysis has presented age and gender adjusted results of the prevalence of PVD ranging from 5.5% to 26.7% (5). In the UKPDS PVD was rare at the time of diagnosis but after 6 years of follow up 11% of the patients had PVD (6). Thus, it is to be noted that PVD is a significant complication of type 2 diabetes

SYMPTOMATIC AND ASYMPTOMATIC PVD :

The symptoms of intermittent claudication is seen only in 50% 0f the patients affected with PVD in this study. It is comparable with other studies, where the prevalence of symptoms among PVD patients was only approximately 50% (2). This may be due to

i. Modern age sedentary life-style.
ii. Many elderly patients not walking far enough to experience symptoms of intermittent claudication.
iii. co-existing neuropathy masking pain sensation.

Since, many of the patients with PVD are asymptomatic it's vital for active screening with ABI in all type 2 Diabetic Subjects.

SEX DISTRIBUTION :

The prevalence of PVD was 20% among males and 14.9% among females in the study group. Diabetes seems to repeal the protective effect of female gender on PVD when compared to non diabetics as seen in other epidemiological studies (3). The higher prevalence among males could be partially due to smoking.

AGING AND PVD :

As the age advances, the prevalence and risk of development of PVD increases and in this study, age adjusted prevalence rates, show a linearly progressive raising trend, and the **highest prevalence was found among the geriatric age group.**

Several epidemiological studies have shown a similar raising trend, proportionate to the advancing age (5, 30, 31). Thus it's to be noted that aging is an unmodifiable risk factor for PVD. And elderly type 2 diabetic patients to be given special attention for PVD prevention and management.

WAIST HIPRATION AND BMI :

Waist hip ratio and BMI were higher for the PVD sub set, indicating that intra abdominal adiposity in general has a important role in cardio-vascular diseases including PVD.

HYPERGLYCAEMIA AND PVD :

As the duration of diabetes increased, the risk of PVD increased in the study group. The large multi centric UKPDS study also has categorically proved that the duration of hyperglycaemia has a definite role in PVD. Fasting plasma glucose was higher among the PVD subset, indicating relatively poor glyceamic control when compared to the rest of the study group. In the large multi centric UKPDS, as the HBA1C increased by every 1 %, there was a increase of 28% in PVD prevalence(6). Tight glycaemic control in general reduces peripheral arterial disease and risk of amputation.

HYPERTENSION AND PVD

Hypertension associated with diabetes raised the risk of PVD, nearly three fold in the study subjects. Another interesting observation noted in the study group was that the duration of hypertension also significantly correlated with the risk of PVD. In the cardiovascular health study hypertensive patients had a 50% increased risk of PVD, in a

multivariate analysis adjusted for age, smoking, diabetes and dyslipideamia (18).

SMOKING AND PVD :

In the study group, smokers had more than four fold increased risk of having PVD, compared to non-smokers. Thus smoking was found to be the single most important risk factor in comparison to other risk factors. A multivariate analysis by kannel, identified smoking as the strongest risk factor for development of PVD (16)

In this study, smoking in a diabetic hypertensive exponentially increased the risk of PVD. Smoking is the single most modifiable risk factor and hence smokers must be actively counselled for cessation of smoking.

DYSLIPIDAEMIA AND PVD :

In the study group, LDL cholesterol and serum triglycerides was relatively higher for PVD subset and the HDL cholesterol was low. The cardiovascular health study and the Edinburg artery study reported higher prevalence of PVD in association with higher LDL, and lower HDL cholesterol in multiple logistic regression analysis (17, 18).

Thus, it's evident that even relatively mild increase in LDL – cholesterol, coupled with high serumtriglycerides and low HDL – cholesterol raises the risk of PVD. Hence, management of atherogenic dyslipidaemia is vital, and lipid levels to be maintained within optimal cut-off limits.

CAD AND PVD :

Type 2 Diabetic patients are more prone to have silent CAD as well as PVD. Co-morbid coronary artery disease had a higher concordance rate (>50%) in the study group subjects affected with PVD. This indicates that an atherosclerotic disease in peripheral arteries should be considered as an indicator of generalized cardio-vascular disease and active secondary preventive measures to be started for prevention of further cardiovascular events. It is imperative to record the ABI – periodically in all Type 2 diabetics, since an ABI of <1 is an CAD equivalent. This assumes importance since many of the patients with ABI < 1 are asymptomatic.

DIABETIC NEPHROPATHY AND PVD :

In the present study group, PVD (ABI<1) was present in 75% of the diabetic nephropathy patients. Diabetic nephropathy was the single most important diabetic microvascular complication associated with

development of PVD. The development of micro albuminuria is a marker of wide spread endothelial dysfunction and development of established diabetic nephropathy leads to more accelerated atherosclerosis, due to associated unfavourably altered lipid profile, hypertension and procoagulant state.

ASSOCIATION OF PVD WITH OTHER COMPLICATIONS OF DIABETES

The prevalence of PVD was 42.5% among patients with diabetic retinopathy in the study group and this under scores the importance of screening for PVD among patients with diabetic retinopathy. Since due to poor visual acuity these patients are more prone for trauma to feet, while walking.

25% of patients with PVD had concomitant co-morbid cerebro vascular disease and this indicates that PVD patients also harbour significant generalized atherosclerosis in other vascular territories as well.

SUMMARY OF THE STUDY

1. Prevalence of peripheral vascular Disease in the study was 17.9% among Type2 Diabetes patients.

2. Prevalence of PVD was 20% among males and 14.9% among females.

3. There was a linear progressive raise in PVD prevalence as age advanced, in the study group subjects.

4. 50% of the patients with PVD are totally asymptomatic.

5. Waist – Hip ratio and BMI were significantly higher for the PVD subset.

6. As the duration of diabetes increased, the risk of PVD increased. There was relatively poor glycaemic control in the PVD sub-set.

7. Hypertension, when associated with Type2 Diabetes raised the risk of PVD nearly three fold. The duration of Hypertension also significantly correlated with the risk of PVD.

8. Smokers had more than four fold increased risk of PVD. Smoking was found to be the single most important independent risk factor for development of PVD.

9. There was relatively higher LDL – Cholesterol, low HDL-Cholesterol, and high Serum Triglycerides in the PVD sub-set.

10. Co-Morbid Coronary artery Disease was widely prevalent (about 60%) among the PVD sub-group (ABI < 1)

11. Diabetic Nephropathy patients had a significantly higher prevalence of PVD.

CONCLUSION

1. **Prevalence of peripheral vascular disease is about 18%** in the present study. This has to be viewed seriously considering the huge type 2 diabetic population. Thus a significant proportion of type 2 diabetic subjects are affected by PVD, and hence due importance to be given for screening and prevention of PVD among type 2 diabetes patients.

2. **About 50% of the PVD patients are totally asymptomatic** and hence the need for active screening with estimation of ABI is to be done annually for all type 2 diabetes patients. This is important for prevention of lower extremity amputation.

3. **Central obesity, Uncontrolled hyperglycemia, hypertension, high LDL cholesterol, high tri glycerides, low-HDL cholesterol and smoking are the modifiable risk factors** associated with development of PVD. **Advancing age** and male gender were found to be the non modifiable risk factors for development of PVD.

4. Concordance rate for co-morbid CAD was very high (>50%) in PVD patients and hence active screening for CAD in all the PVD patients has to be done, even if there is no CAD symptoms.

5. PVD has to be given due importance, and ABI has to be estimated in all type 2 diabetic patients. Low ABI is associated with cardiovascular complications.

Thus, **ABI is a good indicator of underlying complications of diabetes mellitus, particularly CAD. ABI estimation is a non invasive cheap, bed-side, and rapid test with a high degree of validity and predictive power and which does not need specially trained persons or costly equipments. Hence, ABI estimation should be done for all diabetic patients annually.**

REFERENCES

1. Joslin's Diabetes Mellitus – 14th Edition, Editors C. Ronald Kahn, Gordon C. Weir, et al.,
2. International Text book of Diabetes Mellitus – 3rd Edition Editors R.A. Defronzo, E.Ferrannini et al.
3. Diabetes and cardio vascular disease – 2nd Edition Edited by Michael T. John Stone, Aristidis veves.
4. Beckman JA, et al., Diabetes and athero sclerosis J AMA 2002; 287; 2570-81.
5. Macgregor A5 et al Role of Systolic – Blood Pressure and Plasma triglycerides in Diabetic peripheral arterial disease. The Edinburgh artery study. Diabetes care 1999; 22; 453-8.
6. Adler AI, et al – UKPDS 59; Hyperglycaemia and other potentially modifiable risk factors for peripheral vascular disease in type 2 Diabetes melitus; Diabetes care 2002; 25; 894-9.
7. Hirsch AT et al, Peripheral arterial disease detection, awareness and treatment in primary care. JAMA 2001; 286; 1317-24.
8. A pelquist J et.al., what is the most effective way to reduce incidence of amputation in the diabetic foot? Diabetes metob res rev 2000; 16 (supl 1); s75-83.
9. Mangement of peripheral arterial disease transatlantic inter-society consensus (TASC). Eur. J. Vasc. Endo vasc Surg, 2000; 19 (supl. A) : si - xxviii, s1-250.
10. Reiber GE et al risk factors for amputation in patients with Diabetes mellitus, a case – control study. Ann intern med 1992; 117; 97-105.

11. Armstrong DG et al, validation of a diabetic wound classification system. Diabetes care 1998; 21; 855-9.
12. Criqui MH et al mortality over a period of 10 years in patients with peripheral arterial disease N.E.J.M. 1992; 326; 381-6.
13. Murabito JM et al. prevalence and clinical correlates of peripheral arterial disease in the Framingham offspring study Am. Heart J 2002; 143; 961-965.
14. Fowkes fg; Epidemiology of peripheral vascular disease. Atherosderosis 1997; 131. (suppl); s29-s31.
15. Meijer WT et al, peripheral arterial disease in the elderly: the Rotterdam study. Arterio sclera thromb vasc biol. 1998; 18; 185-192.
16. Kannel WB, et al. A general Cardiovascular risk profile ; the Framingham study. Am. J cardiology 1976; 38(1): 46-51.
17. Fowkes FG etal smoking, lipids glucose tolerance, and Blood pressure as risk factors for peripheral atherosclerosis in the edinburg artery study. Am.j. Epidemiology. 1992; 135(4) : 331-340.
18. Newman. AB.et al. Ankle – Arm Index as a marker of atherosclerosis in the cardiovascular health study. Circulation. 1993; 88: 837-845.
19. Bainton D, et al. peripheral vascular disease consequence for survival and association with risk factors in the speed well prospective heart disease study. Br.Heart. J. 1994; 72(2); 128-132.
20. Allan PL et al. Relationship between arotid intima – media thickness and symptomatic and asymptomatic peripheral arterial disease. The Edinburgh Artery study. Stroke 1997; 28(2) : 348-353.
21. Strandness DE et al, combined clinical and pathologic study of diabetic and non-diabetic peripheral arterial disease, Diabetes 1964; 13; 366-372.

22. Barner HB et al. Blood flow in the diabetic leg. Circulation 1971; 43: 391-394.
23. Price JF et al. Relationship between smoking and cardiovascular risk factors in the development of peripheral arterial disease and coronary artery disease; Edinburgh artery study. Eur. Heart- J 1999; 20 : 344-53.
24. O'Neal DN, et al. Lipid levels and peripheral vascular disease in diabetic and non-diabetic subjects. Atherosclerosis 1998; 136:1-8.
25. Schaper NC et al., peripheral vascular disease and type 2 diabetes mellitus. Diabetes metab res Rev.2000; 16 (supp 1) ; s11-5.
26. Taylor Lm Jr et al. The association of elevated plasma homocysteine with progression of symptomatic peripheral arterial disease. J. vasc. Surg. 1991; 13:128-36.
27. Takolander R et al. The use of non-invasiveVascular assessment in diabetic patients with foot lesions. Diabet med 1996; 13:s39-42.
28. Ouriel K etal., peripheral arterial disease lancet 2001; 358:1257-64.
29. Orchard TJ etal. Assessment of peripheral vascular disease in diabetes. Circulation 1993; 88: 819-28.
30. Beach Kw etal, progression of lower extremity arterial ocelusive disease in type II diabetes mellitus. Diabetes care. 1988; 11:464-72.
31. Beks PJ etal peripheral arterial disease in relation to glycaemic level in an elderly Caucasian population: the hoorn study. Diabetologia 1995 : 38: 86-96.
32. Mohan V, Premalatha G, Shastry NG. Peripheral vascular disease in non-insulin dependent diabetes mellitus in South India; Diabetes Research and Clinical Practice 27 (1995); 235-40.

33. Pendsey SP. Peripheral vascular disease. In Practical Management of Diabetes. Editor Pendsey SP, Jaypee Brothers, 1997; 26 :144 - 51.
34. Raman PC, Bhagwat A, Ankle Brachial Index in peripheral vascular disease in Diabetes Mellitus. JAPI, 1997; 45 : 6, 440 – 2.

A STUDY ON PREVALENCE AND RISK FACTORS ASSOCIATED WITH PVD IN TYPE 2 DIABETES

CASE PROFORMA

I a) Name b) Age/Sex | | | c) DOB

d) OP No e) Diabetology No

II i) History of Present illness :- (With special reference to PVD)

ii) | Yes | No | Symptoms of PVD

iii) Past History:-

iv) Smoking | Yes | No | PACK YEARS

Cigarettes	Beedi

To Bacco CHEWER / SNUFF USER
SUBSTANCE ABUSER

v) | Yes | No | Alcoholism

III)

a) Type of DIABETES

Type 1	Type 2	MODY	Secondary Diabetes

b) Duration of Diabetes ---------
(Since Diagnosis)

< 5 years	5-10 Years	>10 Years	>15 years

c) GLYCEAEMIC STATUS

Fasting<140 mg	>140-<170 mg	>170 mg

IV) a) Ht.----Cm ----Wt -------Kg BMI-------

Normal	Over Wt	Obesity

b) WHR ---------

Normal	Central Obesity

V a) [ ] BP

b)

Yes	No

HYPERTENSION.

c) Duration of Hyper tension (Years) [ ]

<5 Years	<10 Years	>10 Years

d) Number of Drugs used for Hypertension Control

1	2	3	3+

VI) LIPID STATUS

a) LDL

<100	100-130	>130

b) HDL

Men	<40	≥40
Women	<50	≥50

c) TRIGLYCERIDES

<150	>150	>400

d) TOTAL CHOLESTEROL

<200	<240	>240

VII

Yes	No

DYSLIPIDAEMIA

VIII a) Peripheral Pulses

	Rt	Lt
DP		
PT		

b) Physical Fitness for ambulation

Yes	No

c) Symptoms of Intermittent claudication

Yes	No

d)

Yes	No

PVD

e) ABI Rt Lt

(By Doppler)

IX) MACRO PROTEINUREA

Yes	No

a)

Urine Albumin	
Blood Urea	
Srcreatinine	

X)

Yes	No

ON ASPIRIN

XI) COMORBID DISEASES

Haematologic Diseases	
Thyroid	
CAD, Cevd	
Neuropathy	
Retinopathy	
Nephropathy	

XII) a) Presence of Mechanical foot Deformities

Yes	No

b) Presence of Foot Infection

Yes	No

c) presence of Non-infected Trophiculcer

Yes	No

d) Duration
_____Month/year

XIII). INVESTIGATIONS

1. Urine Albumin Deposits

2. Hb% TC, DC, ESR

3. Plasma

F	PP

Glucose

4. Blood Urea

5. Serum Creatinine

6. Lipid Profile

TC	LDL	HDL	TGL	VLDL

7. E.C.G

8. Doppler Study of Lower Limb Arteries

MASTER CHART

S.No	Age	Sex	BMI	WHR	Smoking in pack years	Duration of DM	Duration of HT	Symptoms	FPG	Urine Albumin
1	62	Male	26	0.94	Non smoker	2	No HT	AS	122	nil
2	48	Male	27	1	Non smoker	Newly detected	Newly detected	AS	140	nil
3	67	Female	26	0.91	Non smoker	Newly detected	Newly detected	SYMP.	132	Yes
4	43	Female	25	0.89	Non smoker	1	1yrs	AS	140	nil
5	41	Female	25	0.92	Non smoker	Newly detected	No HT	AS	199	nil
6	38	Female	24	0.84	Non smoker	2	Newly detected	AS	153	nil
7	60	Male	26	0.95	18	Newly detected	Newly detected	AS	88	trace
8	46	Female	25	0.96	Non smoker	2	No HT	AS	246	nil
9	54	Female	25	0.89	Non smoker	Newly detected	10	AS	153	nil
10	60	Female	26	0.97	Non smoker	Newly detected	Newly detected	SYMP.	226	nil
11	50	Male	24.5	0.95	15	11	11yrs	AS	206	nil
12	53	Female	26	0.86	Non smoker	Newly detected	No HT	AS	140	nil
13	69	Male	27	0.9	Non smoker	1	No HT	AS	160	Yes
14	53	Male	27.4	1	1Non smoker	12	No HT	AS	170	nil
15	41	Male	26	0.9	Non smoker	5	No HT	AS	170	nil
16	63	Male	23	0.8	16	10	No HT	AS	185	nil
17	44	Female	29	1.05	Non smoker	1	No HT	AS	170	nil
18	51	Male	24.5	0.92	Non smoker	11	Newly detected	AS	170	trace
19	56	Male	27	0.96	Non smoker	3	No HT	AS	111	nil
20	47	Male	24.5	0.91	Non smoker	5	Newly detected	AS	176	Yes
21	47	Male	28	1.12	8	5	3months	AS	212	trace
22	64	Male	20.2	0.8	16	1.5	No HT	AS	216	trace
23	62	Female	27	1.12	Non smoker	20	18	AS	198	trace
24	42	Male	21.5	0.75	10	2	No HT	AS	198	nil
25	31	Female	33.2	1.12	Non smoker	3	Newly detected	AS	140	nil
26	47	Male	27	1.05	14	1.5	No HT	AS	182	trace
27	43	Female	28	0.95	Non smoker	3	3yrs	AS	255	nil
28	63	Male	21.5	0.78	Non smoker	4 months	No HT	AS	152	nil
29	62	Female	27	0.92	Non smoker	Newly detected	3yrs	AS	262	nil
30	50	Female	19	0.72	Non smoker	7	7yrs	AS	220	nil
31	52	Female	21	0.7	Non smoker	13	Newly detected	AS	186	nil
32	46	Female	23	0.78	Non smoker	8	No HT	AS	146	nil
33	66	Male	21	0.8	35	10	1yrs	SYMP.	290	nil

S.No	Age	Sex	BMI	WHR	Smoking in pack years	Duration of DM	Duration of HT	Symptoms	FPG	Urine Albumin
34	65	Female	26	0.82	Non smoker	1	2 yrs	AS	110	nil
35	42	Male	24	0.86	10	2	No HT	AS	110	nil
36	50	Female	30.5	1.01	Non smoker	2	2 yrs	AS	168	nil
37	42	Female	21	0.77	Non smoker	3	No HT	AS	200	nil
38	52	Male	28.5	0.96	Non smoker	Newly detected	Newly detected	AS	152	nil
39	42	Male	29	1.16	16	Newly detected	No HT	AS	205	nil
40	36	Male	24	0.8	Non smoker	1	1yrs	AS	168	nil
41	34	Male	27	0.96	8	Newly detected	No HT	AS	176	nil
42	55	Female	24	0.8	Non smoker	6months	No HT	AS	180	nil
43	45	Female	23	0.8	Non smoker	3	3yrs	AS	150	nil
44	74	Female	31	0.96	Non smoker	1	1yrs	AS	178	nil
45	41	Male	26.5	0.95	16	Newly detected	7yrs	AS	186	nil
46	55	Male	22.5	0.8	Non smoker	5	Newly detected	AS	182	nil
47	45	Male	24	0.88	20	Newly detected	No HT	AS	196	nil
48	32	Female	24	0.81	Non smoker	Newly detected	No HT	AS	146	nil
49	40	Female	24	0.85	Non smoker	2	No HT	AS	146	nil
50	42	Female	25	0.86	Non smoker	4	4	AS	115	trace
51	52	Male	24	0.88	8	10	No HT	AS	149	nil
52	56	Male	25	0.9	Non smoker	10	10	AS	156	nil
53	52	Male	26	0.96	Non smoker	12	7	AS	167	Yes
54	58	Female	24	0.8	Non smoker	12	No HT	AS	108	trace
55	46	Male	24	0.88	Non smoker	8	6	AS	118	nil
56	38	Male	25	0.77	Non Smoker	4	4	AS	146	nil
57	60	Male	25	0.85	Non smoker	12	6	AS	168	trace
58	48	Male	26	0.88	Non smoker	12	10	AS	166	nil
59	52	Male	24	0.9	Non smoker	12	No HT	AS	110	nil
60	58	Male	27	1.01	Non smoker	16	16	SYMP.	178	trace
61	54	Female	24	0.81	Non smoker	11	11	AS	128	nil
62	58	Male	24	0.9	12	10	No HT	AS	136	nil
63	36	Male	25	0.81	Non smoker	2	No HT	AS	106	nil
64	56	Female	25	0.88	Non smoker	11	6	AS	146	nil
65	48	Male	24	0.9	Non smoker	8	No HT	AS	138	nil
66	62	Female	26	0.78	Non smoker	14	No HT	AS	96	trace
67	64	Male	24	0.81	Non smoker	20	No HT	AS	168	nil
68	62	Female	25	0.9	Non smoker	20	15	SYMP	148	Yes

S.No	Age	Sex	BMI	WHR	Smoking in pack years	Duration of DM	Duration of HT	Symptoms	FPG	Urine Albumin
69	56	Female	24	0.84	Non smoker	12	8	AS	116	nil
70	52	Male	24	0.84	Non smoker	10	No HT	AS	98	nil
71	48	Male	23	0.7	Non smoker	8	No HT	AS	141	nil
72	62	Male	26	0.86	15	10	No HT	AS	156	nil
73	39	Male	26	0.86	Non smoker	4	No HT	AS	106	nil
74	74	Female	26	0.84	Non smoker	14	No HT	AS	138	trace
75	68	Female	26	0.82	Non smoker	18	10	AS	136	nil
76	68	Male	26	0.95	10	28	28	AS	158	nil
77	59	Male	23	0.8	Non smoker	13	13	AS	106	nil
78	72	Male	25	0.88	Non smoker	20	20	AS.	162	Yes
79	38	Male	23	0.75	Non smoker	2	No HT	AS	116	nil
80	55	Male	26	0.9	18	15	No HT	SYMP.	220	nil
81	42	Female	22	0.75	Non smoker	6	No HT	AS	126	nil
82	36	Female	23	0.77	Non smoker	6	No HT	AS	136	nil
83	48	Female	24	0.81	Non smoker	8	8	AS	146	trace
84	46	Male	24	0.81	8	6	No HT	AS	148	trace
85	50	Male	25	0.86	Non smoker	10	No HT	AS	128	nil
86	62	Female	26	0.87	Non smoker	17	12	SYMP.	168	nil
87	52	Male	24	0.87	Non smoker	12	No HT	AS	156	nil
88	56	Male	24	0.9	Non smoker	16	6	AS	116	nil
89	60	Male	26	0.9	Non smoker	15	15	AS	126	trace
90	58	Female	26	0.78	Non smoker	13	No HT	AS	116	nil
91	64	Male	25	0.92	20	14	14	SYMP.	168	nil
92	62	Female	25	0.78	Non smoker	17	No HT	AS	128	nil
93	64	Male	25	0.84	Non smoker	19	10	AS	136	trace
94	72	Male	23	0.8	Non smoker	12	No HT	AS	148	nil
95	66	Male	23	0.77	Non smoker	16	16	AS	148	nil
96	65	Male	26	0.98	20	11	11	SYMP.	168	Yes
97	56	Female	24	0.88	Non smoker	16	No HT	AS	146	nil
98	62	Female	24	0.88	Non smoker	14	No HT	AS	156	nil
99	55	Male	25	0.86	12	12	12	AS	156	trace
100	34	Male	25	0.86	Non smoker	6	6	AS	152	nil
101	55	Male	23	0.78	12	10	10	AS	148	nil
102	58	Male	26	1.01	16	11	4	SYMP.	178	Yes
103	54	Female	23	0.78	Non smoker	10	No HT	AS	161	nil

S.No	Age	Sex	BMI	WHR	Smoking in pack years	Duration of DM	Duration of HT	Symptoms	FPG	Urine Albumin
104	70	Male	21	0.75	Non smoker	14	No HT	AS	162	trace
105	59	Female	20	0.72	Non smoker	12	8	AS	158	nil
106	66	Female	25	0.92	Non smoker	12	No HT	AS	188	nil
107	54	Female	20	0.7	Non smoker	12	No HT	AS	116	trace
108	62	Male	20	0.7	Non smoker	12	10	AS	96	trace
109	65	Female	26	0.91	Non smoker	12	No HT	AS	148	nil
110	66	Female	21	0.7	Non smoker	16	12	AS	106	nil
111	80	Male	24	0.88	26	15	15	SYMP.	128	nil
112	60	Male	25	0.91	18	12	No HT	AS	112	nil

MASTER CHART (continued)

S.No	LDL	HDL	TGL	Sys.arm	Dias arm	Ank.sys. RT	Ank.sys. Left	ABI Rt	ABI Lt	Least ABI	PVD	Blood urea	Serum creat.	CEVD	CAD	Nephropathy	Fundus	Neuropathy
1	164	35	140	120	80	120	120	1	1	1	No	19	0.8	No	Yes	No	Normal	No
2	115	64	84	150	90	150	150	1	1	1	No	28	0.9	No	No	No	Normal	No
3	152	43	302	220	110	140	130	0.6	0.6	0.59	Yes	60	1.3	No	No	yes	Abnormal	Yes
4	120	39	262	150	100	140	140	0.9	0.9	0.93	Yes	20	1	No	No	No	Normal	Yes
5	142	32	128	130	90	130	130	1	1	1	No	20	0.8	No	No	No	Abnormal	No
6	85	38	100	150	80	150	170	1	1.1	1	No	30	1	No	No	No	Normal	Yes
7	92	52	138	150	90	160	170	1.1	1.1	1.07	No	17	0.8	No	No	No	Abnormal	Yes
8	87	33	180	130	80	150	150	1.2	1.2	1.15	No	18	0.7	No	No	No	Normal	Yes
9	118	47	114	170	120	200	170	1.2	1	1	No	18	0.7	No	No	No	Normal	Yes
10	105	51	146	150	100	160	170	1.1	1.1	1.07	No	19	0.8	No	No	No	Normal	Yes
11	130	30	290	140	84	160	160	1.1	1.1	1.14	No	28	1.3	No	Yes	No	Normal	Yes
12	160	30	240	132	88	142	140	1.1	1.1	1.06	No	30	0.9	No	No	No	Normal	No
13	120	38	180	130	80	140	140	1.1	1.1	1.08	No	38	1.4	No	Yes	yes	Normal	Yes
14	140	30	190	90	60	100	100	1.1	1.1	1.11	No	30	0.9	No	Yes	No	Normal	Yes
15	130	30	160	120	80	130	130	1.1	1.1	1.08	No	32	0.8	No	No	No	Normal	No
16	130	30	160	120	70	132	132	1.1	1.1	1.1	No	30	0.9	No	No	No	Normal	Yes
17	130	36	162	140	90	148	152	1.1	1.1	1.06	No	28	0.8	No	No	No	Normal	Yes
18	132	32	152	150	100	162	160	1.1	1.1	1.07	No	34	0.9	No	No	No	Abnormal	Yes
19	130	28	152	140	90	154	154	1.1	1.1	1.1	No	36	0.9	No	No	No	Normal	No
20	156	28	190	160	90	160	160	1	1	1	No	40	1.3	No	No	yes	Abnormal	Yes
21	148	30	196	190	80	212	206	1.1	1.1	1.08	No	38	1.2	No	No	No	Normal	Yes
22	128	28	166	140	80	156	158	1.1	1.1	1.11	No	30	0.9	YES	No	No	Normal	No
23	126	30	152	190	80	206	204	1.1	1.1	1.07	No	30	0.9	No	No	No	Normal	Yes
24	132	32	162	130	90	144	146	1.1	1.1	1.11	No	30	0.9	NIL	No	No	Normal	Yes
25	136	32	158	130	100	146	146	1.1	1.1	1.12	No	28	0.9	NIL	No	No	Normal	Yes
26	146	30	178	110	90	110	100	1	0.9	0.91	Yes	36	0.9	NIL	AWMI	No	Normal	Yes
27	136	32	250	150	100	164	166	1.1	1.1	1.09	No	32	0.9	NIL	No	No	Abnormal	Yes
28	128	32	150	130	80	146	146	1.1	1.1	1.11	No	28	0.8	NIL	No	No	Normal	No
29	138	38	200	140	90	154	156	1.1	1.1	1.1	No	32	0.9	NIL	IHD	No	Abnormal	Yes
30	108	40	220	140	86	154	154	1.1	1.1	1.1	No	28	0.8	No	No	No	Abnormal	Yes
31	146	28	236	180	100	170	170	0.9	0.9	0.94	Yes	36	0.8	TIA	No	No	Abnormal	Yes
32	136	46	176	140	80	156	156	1.1	1.1	1.11	No	26	0.7	NIL	No	NIL	Normal	Yes
33	142	26	290	120	60	60	60	0.5	0.5	0.5	Yes	36	0.9	No	IWMI	No	Abnormal	Yes

S.No	LDL	HDL	TGL	Sys.arm	Dias arm	Ank.sys. RT	Ank.sys. Left	ABI Rt	ABI Lt	Least ABI	PVD	Blood urea	Serum creat.	CEVD	CAD	Nephropathy	Fundus	Neuropathy
34	100	38	148	180	80	194	196	1.1	1.1	1.08	No	26	0.7	No	No	No	Normal	No
35	116	30	148	110	70	120	120	1.1	1.1	1.09	No	26	0.8	No	No	No	Abnormal	Yes
36	148	30	198	180	110	186	186	1	1	1.03	No	36	0.8	No	No	No	Normal	No
37	116	38	200	110	80	126	124	1.2	1.1	1.13	No	28	0.8	No	IHD	No	Abnormal	Yes
38	138	28	168	130	100	146	146	1.1	1.1	1.11	No	36	0.9	No	No	No	Normal	No
39	146	28	196	130	80	148	146	1.1	1.1	1.11	No	26	0.8	No	No	No	Normal	Yes
40	128	30	162	140	100	156	158	1.1	1.1	1.11	No	28	0.8	No	No	No	Normal	No
41	128	30	176	136	88	126	136	0.9	1	0.93	Yes	28	0.8	YES	No	No	Normal	No
42	110	38	146	140	80	150	150	1.1	1.1	1.07	No	28	0.8	No	No	No	Normal	Yes
43	116	36	156	150	106	158	158	1.1	1.1	1.05	No	29	0.8	No	No	No	Abnormal	Yes
44	136	32	196	210	84	218	220	1	1.1	1.04	No	32	0.9	No	IHD	No	Abnormal	Yes
45	116	32	140	120	100	120	100	1	0.8	0.83	Yes	30	0.8	YES	No	No	Abnormal	No
46	128	30	166	160	106	170	170	1.1	1.1	1.06	No	18	0.7	No	No	No	Normal	No
47	136	32	206	100	70	110	112	1.1	1.1	1.1	No	22	0.8	No	No	No	Normal	No
48	106	40	146	120	80	130	130	1.1	1.1	1.08	No	18	0.6	No	No	No	Normal	No
49	116	46	148	150	100	160	160	1.1	1.1	1.07	No	20	0.7	No	No	No	Normal	Yes
50	112	44	162	150	100	160	162	1.1	1.1	1.07	No	22	0.6	No	No	No	Abnormal	Yes
51	126	30	156	140	90	150	150	1.1	1.1	1.07	No	26	0.7	No	No	No	Normal	Yes
52	128	30	146	160	100	170	172	1.1	1.1	1.06	No	28	0.8	No	IHD	No	Normal	Yes
53	138	30	198	80	100	160	160	0.9	0.9	0.89	Yes	48	1.7	No	IWMI	yes	Abnormal	Yes
54	112	40	140	130	80	140	140	1.1	1.1	1.08	No	28	0.8	No	No	No	Normal	Yes
55	118	40	160	150	98	158	160	1.1	1.1	1.05	No	30	0.7	No	No	No	Normal	Yes
56	128	36	152	150	90	158	160	1.1	1.1	1.05	No	28	0.6	No	No	No	Normal	No
57	131	31	156	130	80	140	140	1.1	1.1	1.08	No	30	0.7	No	No	No	Normal	Yes
58	136	30	164	150	96	160	160	1.1	1.1	1.07	No	28	0.9	No	No	No	Abnormal	Yes
59	106	36	136	120	80	130	130	1.1	1.1	1.08	No	30	0.8	No	No	No	Normal	Yes
60	142	28	188	160	100	100	130	0.6	0.8	0.63	Yes	36	0.8	No	CAD	No	Normal	Yes
61	102	42	142	150	96	160	162	1.1	1.1	1.07	No	26	0.6	No	No	No	Normal	Yes
62	108	40	160	120	80	130	130	1.1	1.1	1.08	No	22	0.7	No	No	No	Normal	Yes
63	98	36	148	120	86	130	132	1.1	1.1	1.08	No	26	0.6	No	No	No	Normal	No
64	96	40	156	140	94	150	152	1.1	1.1	1.07	No	30	0.7	No	No	No	Normal	Yes
65	102	38	142	130	70	140	142	1.1	1.1	1.08	No	26	0.6	No	No	No	Normal	No
66	106	36	136	120	60	130	130	1.1	1.1	1.08	No	28	0.7	No	No	No	Normal	Yes
67	138	28	158	130	70	140	142	1.1	1.1	1.09	No	28	0.8	No	No	No	Normal	Yes
68	136	28	190	160	100	140	100	0.9	0.6	0.63	Yes	55	2.3	No	No	yes	Abnormal	Yes

S.No	LDL	HDL	TGL	Sys.arm	Dias arm	Ank.sys. RT	Ank.sys. Left	ABI Rt	ABI Lt	Least ABI	PVD	Blood urea	Serum creat.	CEVD	CAD	Nephropathy	Fundus	Neuropathy
69	128	38	168	150	86	158	160	1.1	1.1	1.05	No	29	0.8	No	No	No	Normal	Yes
70	96	28	106	126	70	138	136	1.1	1.1	1.08	No	22	0.7	No	No	No	Normal	Yes
71	106	39	162	130	82	140	142	1.1	14	1.08	No	29	0.8	No	No	No	Normal	No
72	142	36	160	128	78	138	140	1.1	1.1	1.08	No	30	0.8	No	ASMI	No	Normal	Yes
73	106	30	148	110	78	122	120	1.1	1.1	1.09	No	26	0.7	No	No	No	Normal	No
74	126	36	156	130	88	140	142	1.1	1.1	1.08	No	32	0.8	No	No	No	Normal	Yes
75	116	26	148	160	106	176	170	1.1	1.1	10.6	No	30	0.8	No	No	No	Abnormal	Yes
76	138	28	186	150	100	100	100	0.7	0.7	0.67	Yes	40	1.2	No	IHD	No	Abnormal	Yes
77	116	36	166	150	80	160	162	1.1	1.1	1.07	No	30	0.8	No	No	No	Normal	Yes
78	136	25	168	150	90	100	90	0.7	0.6	0.6	Yes	40	1.3	No	IHD	yes	Abnormal	Yes
79	126	30	150	130	80	140	142	1.1	1.1	1.08	No	26	0.8	No	No	No	Normal	No
80	142	38	166	140	90	100	100	0.7	0.7	0.71	Yes	32	0.9	No	No	No	Normal	Yes
81	106	30	156	130	70	140	142	1.1	1.1	1.08	No	26	0.7	No	No	No	Normal	No
82	116	36	146	120	86	130	128	1.1	1.1	1.07	No	27	0.8	No	No	No	Normal	Yes
83	118	36	136	160	100	170	168	1.1	1.1	1.05	No	30	0.9	No	No	No	Abnormal	Yes
84	96	32	138	130	86	140	140	1.1	1.1	1.08	No	32	0.8	No	No	No	Normal	Yes
85	108	33	140	138	76	148	150	1.1	1.1	1.07	No	32	0.7	NO	No	No	Normal	Yes
86	136	32	168	160	110	106	110	0.7	0.7	0.66	Yes	28	0.9	TIA	IWMI	No	Normal	Yes
87	116	34	142	120	78	130	130	1.1	1.1	1.08	No	32	0.8	NO	No	No	Normal	Yes
88	120	34	142	150	90	160	162	1.1	1.1	1.07	No	26	0.6	NO	No	No	Normal	Yes
89	108	35	144	150	78	158	158	1.1	1.1	1.05	No	28	0.9	No	No	No	Normal	Yes
90	98	35	146	120	70	136	132	1.1	1.1	1.1	No	28	0.9	No	No	No	Normal	Yes
91	133	29	196	160	110	90	90	0.6	0.6	0.56	Yes	30	0.8	No	ASMI	No	Abnormal	Yes
92	106	35	126	120	70	132	136	1.1	1.1	1.1	No	26	0.7	No	No	No	Normal	Yes
93	116	35	128	140	76	148	150	1.1	1.1	1.06	No	26	0.8	No	IHD	No	Normal	Yes
94	108	36	128	130	80	140	142	1.1	1.1	1.08	No	26	0.9	No	No	No	Normal	Yes
95	108	38	132	160	110	170	174	1.1	1.1	1.06	No	30	0.9	No	IHD	No	Normal	Yes
96	142	26	210	160	100	100	100	0.6	0.6	0.63	Yes	48	1.8	No	IWMI	yes	Abnormal	Yes
97	116	38	160	130	80	140	142	1.1	1.1	1.08	No	30	1	No	No	No	Normal	Yes
98	126	38	161	120	70	132	130	1.1	1.1	1.08	No	30	0.9	No	No	No	Normal	Yes
99	130	38	162	140	88	148	148	1.1	1.1	1.06	No	30	1.1	No	No	No	Normal	Yes
100	136	32	166	160	80	170	174	1.1	1.1	1.06	No	32	1	No	No	No	Normal	No
101	136	32	142	140	88	148	152	1.1	1.1	1.06	No	32	1.2	No	No	No	Normal	Yes
102	152	26	186	190	110	100	90	0.5	0.5	0.47	Yes	56	2.8	NO	IHD	yes	Abnormal	Yes
103	142	26	172	120	70	130	132	1.1	1.1	1.08	No	32	1	NO	No	No	Normal	No

S.No	LDL	HDL	TGL	Sys.arm	Dias arm	Ank.sys. RT	Ank.sys. Left	ABI Rt	ABI Lt	Least ABI	PVD	Blood urea	Serum creat.	CEVD	CAD	Nephropathy	Fundus	Neuropathy
104	136	28	182	140	80	150	150	1.1	1.1	1.07	No	32	1.2	TIA	No	No	Normal	Yes
105	136	32	136	160	106	172	174	1.1	1.1	1.08	No	30	0.9	No	No	No	Abnormal	Yes
106	132	28	190	140	90	120	100	0.9	0.7	0.71	Yes	36	0.8	No	CAD	No	Normal	Yes
107	126	28	140	130	80	142	142	1.1	1.1	10.9	No	30	0.9	No	No	No	Normal	Yes
108	116	32	136	130	98	144	144	1.1	1.1	1.11	No	30	0.9	No	No	No	Normal	Yes
109	128	32	180	130	90	100	100	0.8	0.8	0.77	Yes	31	0.9	No	CAD	No	Normal	Yes
110	116	26	126	140	100	150	154	1.1	1.1	1.07	No	30	1.2	No	No	No	Normal	Yes
111	116	31	140	160	100	60	60	0.4	0.4	0.38	Yes	29	1.2	TIA	No	No	Normal	Yes
112	120	38	130	130	70	142	144	1.1	1.1	1.09	No	26	0.8	NO	No	No	Normal	Yes